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RAPESEED OIL: NUTRITIONAL AND BIOCHEMICAL EFFECTS ON THE CHICK

by

© SHEILA MARGARET WILSON

A THESIS
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ABSTRACT

An experiment was conducted to determine the effects of increasing the level of saturated fatty acids in diets containing rapeseed oils on their nutritive properties for the chick using rate of growth, energetic efficiency, and tissue composition as criteria for comparison. The diets fed contained 20 parts high erucic acid rapeseed oil (HER), low erucic acid rapeseed oil (LER) or sunflowerseed oil (SFO) or 15 parts HER or LER with 5 parts palmitic or oleic acid, and were formulated by isocaloric substitution of the respective oil or oil mixture for glucose.

Results showed that increasing the saturated fatty acid content of diets containing HER by the addition of palmitic acid had no beneficial effect on the decreased weight gain, fat deposition, or energetic efficiency, or heart enlargement observed in chicks fed diets containing 20 parts HER for 3 to 4 weeks, when compared with chicks fed diets containing HER supplemented with oleic acid.

Chicks fed diets containing 20 parts LER for 3 to 4 weeks gained more weight, had smaller hearts, and utilized energy with greater efficiency than chicks fed diets containing HER. However, body fat gain and energetic efficiency of chicks pair-fed diets containing LER for 3 to 4 weeks were lower than of chicks pair-fed diets

containing SFO. Increasing the saturated fatty acid intake of chicks fed diets containing LER improved neither fat deposition nor energetic efficiency.

Analysis of the fatty acid composition of heart and carcass lipid by gas chromatography showed chicks fed a diet containing HER plus oleic acid for 7 days or 3 to 4 weeks deposited less erucic and eicosenoic acid than chicks fed comparable diets containing HER plus palmitic acid.

In vitro studies showed that mitochondria isolated from the heart and skeletal muscle of chicks fed diets containing 20 parts HER for 28 days had reduced ADP/O ratios, but similar rates of oxygen uptake and ATP synthesis, when compared with mitochondria isolated from chicks fed comparable diets containing SFO.

Mitochondria isolated from the heart and skeletal muscle of chicks fed diets containing 20 parts LER for 28 days showed rates of oxygen uptake and ATP synthesis similar to those of chicks fed diets containing SFO. ADP/O ratios of skeletal muscle mitochondria isolated from chicks fed diets containing LER were similar to those of chicks fed diets containing SFO; however, ADP/O ratios of cardiac mitochondria were intermediary between and no different from, those of chicks fed diets containing HER or SFO.

These data suggest that the erucic and eicosenoic

acids present in HER are the prime factor responsible for the poorer nutritive properties of this oil for the chick, and that the low saturated fatty acid content of rapeseed oils do not impair their nutritive value. The presence of erucic acid in the diet, possibly even at such low levels as found in LER, caused reduced mitochondrial oxidative efficiency and may thus explain the decreased energetic efficiency and body fat gain observed in chicks fed diets containing 20 parts HER or LER for 3 to 4 weeks.

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TABLE OF CONTENTS

PAGE

INTRODUCTION 1

LITERATURE REVIEW 2

EXPERIMENT 1. Effects of Saturated Fatty Acid Supplementation of Diets Containing High and Low Erucic Acid Rapeseed Oils for the Chick.

Materials and Methods . . 12

Results 19

Discussion 32

EXPERIMENTS 2 AND 3. Oxidative Capacity of Mitochondria Isolated from the Heart and Skeletal Muscle of Chicks Fed Diets Containing High or Low Erucic Acid Rapeseed Oil, or Sunflower Oil.

Materials and Methods . . 36

Results 41

Discussion 53

SUMMARY 61

BIBLIOGRAPHY 64

APPENDIX 70

Carcass and heart fatty acid compositions (Exp. 1) . . . 71

Carcass and heart fatty acid compositions (Exp. 2) . . . 73

Carcass and heart fatty acid compositions (Exp. 3) . . . 75

LIST OF TABLES

TABLE PAGE

1. Composition of high carbohydrate diet . . 13
2. Composition of diets. 14
3. Fatty acid composition of oils. 18
4. Weight gain, energy consumption and feed efficiency of chicks fed experimental diets ad libitum for 20 or 26 days. . . 20
5. Weight gain, energy consumption and feed efficiency of chicks pair-fed experimental diets for 20 or 26 days. 22
6. Energy consumption, carcass gain of fat and protein and energy utilization of chicks pair-fed experimental diets for 20 or 26 days. 24
7. Weight gain, energy consumption, carcass gain of fat and protein and energy utilization of chicks pair-fed experimental diets for 7 days. 25
8. Weight and fat content of hearts of chicks fed experimental diets for 7 and for 20 or 26 days. 27
9. Erucic acid content of heart and carcass fat of chicks fed experimental diets . . 29
10. Eicosenoic acid content of heart and carcass fat of chicks fed experimental diets. 31

TABLE PAGE

11. Composition of diets 38

12. Fatty acid composition of oils . . 39

13. Weight gain, energy consumption and feed efficiency of chicks pair-fed experimental diets for 7 or 28 days. 42

14. Incidence of hydropericarduim and abnormal liver changes in chicks pair-fed experimental diets for 7 or 28 days. 44

15. Weight of hearts and fat content of heart and skeletal muscle of chicks pair-fed experimental diets for 7 or 28 days. 45

16. Per cent saturated fatty acids, unsaturation index and % n-9 fatty acids of heart lipid from chicks pair-fed experimental diets for 7 or 28 days. . 47

17. Per cent saturated fatty acids, unsaturation index and % n-9 fatty acids of skeletal muscle lipid from chicks pair-fed experimental diets for 7 or 28 days. 48

18. Oxidative activity of cardiac mitochondria isolated in the presence of heparin from chicks fed diets containing SFO, HER or LER for 7 or 28 days. 50

TABLE PAGE

19. Oxidative activity of skeletal muscle mitochondria isolated in the presence of heparin from chicks fed diets containing SFO, HER or LER for 7 or 28 days. . . 52

INTRODUCTION

Studies on the nutritive value of rapeseed oil were intensified in 1970 with the finding that rapeseed oil induced changes in the myocardium. Since that time many studies have been conducted to determine the factor(s) in rapeseed oil responsible for its cardiopathogenicity and its (their) mode of action. Recently the question has been raised as to whether a component(s) in rapeseed oil interferes with energy utilization thus contributing to pathological changes in heart and skeletal muscle. Since only limited information is available on the effect of high and low erucic acid rapeseed oil on energy utilization, the following in vivo and in vitro studies were conducted using the chick as the experimental animal.

LITERATURE REVIEW

The nutritive value of rapeseed oil containing a high level of erucic acid (HER), and that of newer varieties of rapeseed containing little or no erucic acid in the oil (LER), have been studied in a variety of animals. These studies have formed the basis of several comprehensive reviews (Borg,1975; Rocquelin et al., 1971; Vles, 1975).

In early studies on the nutritive value of HER for the chick, Sell and Hodgson (1962) showed that when included at 4 or 8% of the diet HER promoted growth as effectively as similar levels of soybean oil, sunflowerseed oil, or tallow. Later, however, Salmon (1969) observed that chicks fed diets containing 10, 7.5, 5 or 2.5% HER combined with 0, 2.5, or 7.5% soybean oil, respectively, showed depressed growth compared with chicks fed a diet containing 10% soybean oil. More recently, Sheppard et al. (1971) have shown that chicks fed a diet containing 16% HER grew significantly slower than chicks fed a diet containing 16% corn oil. Similarly, Vogtmann et al. (1973), found that incorporation of 15% HER in the diet of the chick consistently produced a lower rate of weight gain than a 15% soybean

oil control diet. Work by Clement (1974) has extended these observations by showing that whilst substitution of 10 parts HER isocalorically for glucose in the chick diet failed to suppress growth, substitution of 20 parts HER caused a significant decrease in growth rate, when compared with a control diet containing sunflowerseed oil. In general, the results of these experiments with the chick are in agreement with results of studies with the rat, which have shown that rats fed diets containing 10% or more HER grow less than rats fed diets containing other dietary oils such as sunflowerseed oil (Abdellatif and Vles, 1970a, 1973), corn oil (Beare et al., 1959; Hornstra, 1972), soybean oil or corn oil (Kramer et al., 1973) and peanut oil (Rocquelin and Cluzan, 1968).

Studies on the nutritional value of LER for the chick have given varying results. Walker et al. (1970) found that chicks fed a diet containing 20% LER (1.2% erucic acid), grew at the same rate as chicks fed a diet containing 20% tallow. In agreement with Walker et al. (1970), Clement (1974) observed a similar rate of weight gain between chicks fed diets containing 10 or 20 parts LER (3.9 - 4.7% erucic acid) and chicks fed diets containing 10 or 20 parts sunflowerseed oil. Sheppard et al. (1971), however, have reported that chicks fed a diet containing 16% LER for 3 weeks grew at a rate which

was significantly slower than chicks fed a diet containing 15% corn oil, and similar to chicks fed a diet containing 15% HER. Vogtmann et al. (1973) observed a variable response to the incorporation of 15% LER (2.8 -2.9% erucic acid) in chick diets; low erucic acid rapeseed oil produced in different instances both depression of, and no effect on, growth when compared with a diet containing 15% soybean oil. In the case of the rat, studies have shown LER to promote as good a body weight gain as sunflowerseed oil (Abdellatif and Vles, 1970a, 1970b), olive oil (Craig and Beare, 1968), soybean and corn oil (Kramer et al., 1973) and peanut oil (Rocquelin and Cluzan, 1968; Rocquelin et al., 1970).

That erucic acid is the primary agent in rapeseed oil responsible for its growth depressing properties has been demonstrated (Abdellatif and Vles, 1973; Rocquelin et al., 1970; Thomasson and Boldingh, 1955). However, it has also been suggested that the poor performance of animals fed diets containing HER may not be due just to the high erucic acid content, but may be due in part to the low level of saturated fatty acids, mainly palmitic acid, giving an unfavorable ratio of saturated to unsaturated fatty acids in this oil (Beare et al., 1963; Kramer et al., 1975; Rocquelin et al., 1971).

In studies with the chick, Clement (1974) showed

that chicks fed diets containing 15 parts HER plus 5 parts palmitic acid grew significantly faster than chicks fed diets containing 20 parts HER, but at a similar rate to chicks fed diets containing 15 parts HER plus 5 parts oleic acid. In studies with the rat, the beneficial effects on growth of increasing the saturated fatty acid content of diets containing HER have been demonstrated in some experiments (Beare et al., 1963), but could not be reproduced in others (Beare-Rogers et al., 1972). Furthermore, LER which has a low palmitic acid content and an unbalanced saturated to unsaturated fatty acid ratio similar to HER, has been shown to produce as good a growth rate as sunflowerseed oil in the chick (Clement, 1974), and as sunflowerseed oil (Abdellatif and Vles, 1970a, 1973) and peanut oil (Rocquelin and Cluzan, 1970) in the rat. No improvement in weight gain of rats was observed when LER was blended with tripalmitin, tristearin, palm oil, or lard (Craig and Beare, 1968). Similarly, Clement (1974), did not find that supplementation of a diet containing LER with palmitic acid increased weight gain of chicks fed ad libitum for 26 days, or body fat gain or energetic efficiency of chicks pair-fed for 26 days. It has been suggested (Rocquelin et al., 1970, 1971) that the observed growth depression in rats fed high erucic acid rapeseed oil is due mainly to the presence

of erucic acid in the diet and that the unbalanced ratio of saturated to unsaturated fatty acids only influence growth when the dietary linoleic acid supply is equal to or less than 10% of the total fatty acid content. Clarification of the possible beneficial effects on growth and energy utilization of supplementing diets containing HER with palmitic acid is required.

In addition to its effects on growth and feed consumption, rapeseed oil feeding has been associated with a variety of pathological changes, predominantly in tissues dependent on fat for energy (Abdellatif and Vles, 1970b, 1973). Marked alteration in the lipid composition of skeletal muscle, heart, liver and other organs has been noted (Abdellatif and Vles, 1970a; Beare-Rogers et al., 1971; Beare-Rogers and Nera, 1972; Kramer et al., 1973; Sell and Hodgson, 1962; Walker, 1970).

It has been established that feeding a diet containing 10% or more HER to the rat is characterized by a rapid rise in cardiac lipid, reaching a maximum intensity after 3-6 days; with continued feeding the lipid level declines towards normal but is followed in 2-6 months by histiocyte infiltration, myocardial necrosis and fibrosis (Abdellatif, 1972; Borg, 1975). Although it is established that the inclusion of LER in the diet of the rat does not lead to an early accumulation of

lipid in the heart, recent evidence has been presented to suggest that the long term feeding of rapeseed oils low in erucic acid content will produce cardiac lesions (Beare-Rogers et al., 1974; Charlton et al., 1975; Rocquelin et al., 1970, 1971). The cause of the cardiac lesions observed when rats are fed diets containing LER for prolonged periods of time is unknown.

The effects of adding saturated fats to diets containing HER or LER on cardiac lipid accumulation and lesions in the rat has not been studied extensively. Beare-Rogers et al. (1972) have reported that rats fed diets containing 20% synthesized oils with a low level of saturated fatty acids and a high level of erucic acid showed histiological indication (Oil Red O or hematoxylin-phloxine-saffron staining) of aggravated deposition of fat and necrosis of the heart, however, addition of saturated fat to the diet had no significant modifying action.

In contrast to the rat, it has been shown that chicks fed a diet containing up to 20 parts HER do not accumulate lipid in their hearts (Clement, 1974; Vogtmann et al., 1974). However, Clement (1974) observed that chicks fed a diet containing 20 parts HER for 24 days had significantly heavier hearts with significantly lower fat content, than chicks fed diets containing 20

parts sunflowerseed oil for 24 days under both pair-feeding and ad libitum regimens. In the same experiment (Clement 1974), the inclusion of 20 parts LER in the chick's diet did not alter heart size or fat content. In a subsequent experiment Clement (1974) found that supplementation of a diet containing HER with palmitic acid significantly decreased heart size after 26 days of ad libitum feeding when compared to a diet containing unsupplemented HER or HER supplemented with oleic acid. Other workers, however, have reported that chicks fed a diet containing 5, 10 or 15% HER, LER or soybean oil for 28 days (Vogtmann et al., 1974), or 16% HER, LER or corn oil for 21 days (Sheppard et al., 1971), had similar heart sizes. The reason for the lack of agreement on the effects of HER on chick heart size between the results of Clement (1974), and those of Vogtmann et al. (1974) and Sheppard et al. (1971) may be due to the higher level of HER in the diets in Clement's study. The reason why, in contrast to the rat, the inclusion of 20 parts HER in the diet of the chick caused an increased heart size but no increase in heart lipid content (Clement, 1974) is not known.

Many studies have been conducted to determine why fat accumulates in the hearts of rats fed diets

containing high erucic acid rapeseed oil. It has been attributed to slower rate of oxidation of erucic acid due to reduced activity of enzymes of fatty acid activation (Blond et al., 1975; Cheng and Pande, 1975); to reduced activity of enzymes of fatty acid oxidation (Kramer et al., 1973; Swarttouw, 1974); to inhibition of mitochondrial oxidation of fatty acids by a mitochondrial metabolite of erucic acid (Christopherson and Bremer, 1972; Christopherson and Christiansen, 1975; Heijkenskjöld and Ernster, 1975); to difficulties in the oxidation of erucic acid involving its carnitine-dependent transport to the sites of β-oxidation (Bulhak-Jachymczyk and Hübner-Wozniak, 1974), and to impairment of ATP utilization due to changes in membrane fatty acid composition (Blomstrand and Svensson, 1974, 1975; Clandinin, 1976b).

Other studies have also indicated that energy utilization may be impaired when rats are fed diets containing high erucic acid rapeseed oil. Thus, Hornstra (1972) found that rats fed diets containing 31% HER gained less weight per unit of digestible energy consumed and had a greater oxygen uptake and water vapor loss than rats fed comparable diets containing sunflowerseed oil. Hornstra (1972) proposed that incorporation of HER in the rat's diet caused a slight uncoupling of oxidative phosphorylation with energy lost in the form of heat rather than conserved as ATP.

Studies by Houtsmuller et al. (1970) have also indicated impaired energy utilization in rats fed diets containing HER. These workers (Houtsmuller et al., 1970) have demonstrated decreased rates of oxygen uptake and ATP synthesis by mitochondria isolated from the hearts of rats previously fed diets containing HER, and thus have proposed that erucic acid causes a decrease in the capacity of isolated heart mitochondria to oxidize substrates. Recently, however, Dow-Walsh et al. (1975) concluded that when isolated in the presence of heparin, mitochondria from the hearts of rats fed a diet containing 20% HER were functionally intact with respect to oxidation and energy coupling capacity .

In in vivo studies, Clement (1974) has shown that chicks fed diets containing 20 parts HER for 26 days utilized energy less efficiently (determined as energy consumed/unit of energy gained) than chicks pair-fed comparable diets containing LER or sunflowerseed oil. In a subsequent experiment, Clement (1974) found that when pair-fed, chicks fed diets containing 15 parts HER plus 5 parts palmitic acid utilized energy more efficiently than chicks fed diets containing 20 parts HER, but with the same efficiency as chicks fed diets containing 15 parts HER plus 5 parts oleic acid. In the

case of LER, Clement (1974) found that chicks fed diets containing 20 parts LER, 15 parts LER plus 5 parts palmitic acid or 15 parts LER plus 5 parts oleic acid showed similar energetic efficiencies (kcal consumed/kcal gained).

The following study was conducted to confirm the aforementioned results of Clement's on the effect on the energetic efficiency of the chick of supplementing a diet containing 15 parts HER or LER with 5 parts palmitic or oleic acid. Subsequently, experiments were conducted to study the oxidative activity of mitochondria isolated from the heart and skeletal muscle of chicks fed diets containing HER, LER or sunflowerseed oil.

EXPERIMENT 1

The purpose of this experiment was to confirm the results of a previous study (Clement, 1974) conducted in this laboratory on the effects on growth and energy utilization of modifying the ratio of saturated to unsaturated fatty acids in high and low erucic acid rapeseed oil, when fed to chicks.

Materials and Methods

Diets containing 20 parts high erucic acid rapeseed oil (HER), 20 parts low erucic acid rapeseed oil (LER), 20 parts sunflowerseed oil (SFO) or mixtures composed of 15 parts HER or LER and 5 parts palmitic or oleic acid were formulated from the high carbohydrate diet (Table 1), by substituting 20 parts oil or oil mixture isocalorically for glucose. Metabolizable energy values used in formulating the chick diets were glucose 3.64 kcal/g (Hill et al., 1960), HER 7.37 kcal/g, LER 8.79 kcal/g, and SFO 8.88 kcal/g(Renner, 1967), palmitic acid 4.59 kcal/g and oleic acid 8.31 kcal/g (Renner and Hill, 1961). The composition of the diets is shown in Table 2.

Each diet was fed ad libitum to duplicate groups

TABLE 1

Composition of high carbohydrate diet

Ingredients	Per cent
Constants	
Soybean meal (50% protein)	35.00
Glycine	1.00
Methionine	0.50
Brewer's dried yeast	2.50
Dried whey	2.00
Limestone	1.14
Dicalcium phosphate	1.84
Sodium chloride	0.60
Soybean oil	0.50
Choline chloride (50%)	0.60
Chromic oxide "bread"[1]	1.00
Mineral mixture[2]	0.41
Vitamin mixture[3]	0.28
Antioxidant[4]	0.025
Variable	
Glucose[5]	52.605

[1] Contains 30% Cr_2O_3 in wheat flour.

[2] Mineral mixture supplied in mg/313 kcal of diet: K_2HPO_4, 220; $MgSO_4$, 115; $MnSO_4 \cdot H_2O$, 33.5; $FeSO_4 \cdot 7H_2O$, 28; $ZnCO_3$, 9.7; $CuSO_4 \cdot 5H_2O$, 0.78; KI, 0.29; $NaSeO_3$, 0.002.

[3] Vitamin mixture supplied in mg/313 kcal of diet: thiamine HCl, 1.0; riboflavin, 1.0; Ca pantothenate, 4.0; biotin, 0.04; pyridoxine, 2.0; niacin, 8.0; folacin, 0.3; menadione, 0.3; aureomycin, 1.0; vitamin B_{12}, 0.000005; vitamin A, 1000 I.U.; vitamin D, 150 I.U.; vitamin E, 3.3 I.U.

[4] Contains 25% ethoxyquin. Monsanto Chemical Co., St. Louis, Missouri, USA.

[5] Cerelose.

TABLE 2

Composition of diets

	Level			Constant ingredients	Glucose[1]	Cellulose[2]	Total
	Oil	Palmitic acid	Oleic acid				
	g	g	g	g	g	g	g
SFO[3]	20	-	-	47.40	3.82	9.44	80.66
HER[4]	20	-	-	47.40	12.11	9.44	88.95
HER	15	5	-	47.40	15.92	9.44	92.76
HER	15	-	5	47.40	10.81	9.44	87.65
LER[5]	20	-	-	47.40	4.31	9.44	81.15
LER	15	5	-	47.40	10.08	9.44	86.92
LER	15	-	5	47.40	4.97	9.44	81.81

[1] Cerelose.

[2] Alpha-floc BW-40. Brown Company, Berlin, New Hampshire, USA.

[3] Sunflowerseed oil, "Safflo." Co-op Vegetable Oils Ltd., Altona, Manitoba.

[4] High erucic acid rapeseed oil. Western Canadian Seed Processors Ltd., Lethbridge, Alberta.

[5] Low erucic acid rapeseed oil. Western Canadian Seed Processors Ltd., Lethbridge, Alberta.

of 10 male crossbred (Dominant White X White Plymouth Rock) chicks from 4 to 24 days of age. In addition, each diet was pair-fed to duplicate groups of chicks from 4 to 11 and from 4 to 24 days of age. In pair-feeding, the feed intake of each group was restricted to that of the chicks fed the diet containing 20 parts HER.

During the first four days of life the chicks were fed a semi-purified, high carbohydrate diet. They were then assigned to the experimental groups, equalizing both mean body weight and weight distribution among the groups. The chicks were housed in electrically heated, thermostatically controlled battery brooders with raised wire-screen floors, in a temperature controlled laboratory.

Water was supplied ad libitum and feed wastage was determined daily throughout the experiment. Data on growth and feed consumption were obtained weekly. During the third week of the experiment excreta were collected from chicks fed ad libitum so that metabolizable energy of the diets could be determined. Collections were made at 24 hour intervals on 3 successive days, and the excreta were kept frozen until processed. Chromic oxide was incorporated into all diets as an index substance, thereby eliminating the need for quantitative measurement of feed intake and

quantitative collection of excreta. The methods for processing excreta, conducting chemical analyses on excreta and diet samples for moisture, nitrogen, combustible energy, fat and chromium oxide, and for computing metabolizable energy have been described previously (Hill and Anderson, 1958, Hill et al., 1960; Renner and Hill, 1960).

At the end of each feeding regimen the chicks were killed using chloroform, and the liver and hearts removed and stored at -29° until analyzed. The contents of the gastro-intestinal tracts were removed and the remaining carcasses of each group were frozen, ground, mixed and a sample dried by lyophilization. The dried samples were then ground in an analytical mill (Teckmar Company, Cincinnati, Ohio, USA). At the beginning of the experiment, 2 groups of 10 four-day-old chicks were killed and prepared for analysis using the above procedure, so that tissue gains of fat and protein of the fed chicks could be calculated.

In preparation for analysis, frozen hearts were sliced thinly and dried by lyophilization. Moisture content was determined by difference. The dried samples were ground in an analytical mill and stored a -29° until analyzed.

Carcass and heart samples of chicks pair-fed for 7 and for 20 days were analyzed for protein, fat, and moisture using the methods described by Hill and

Anderson (1958). The fatty acid composition of heart and carcass lipid were determined from methyl esters, prepared by the method of Metcalfe et al. (1966) and using a gas chromatograph (Bendix 2500, Model 2532-2, Bendix Process Instr. Div., Ronceverte, West Virginia, USA),equipped with a 350 X 4mm glass column packed with 10% phenyl-50 cyanopropyl on 80/100 mesh Chromosorb W and a flame ionization detector. Peaks were quantitated by use of a digital integrator (model 5300, Spectra Physics, Santa Clara, California) and identified by comparison with methyl esters of known reference oils (AOCS oil reference mixture RM-3, Supelco Inc., Bellefonte, Pennsylvania).

The fatty acid composition of the oils fed was determined by the methods used for analysis of heart and carcass lipid. The fatty acid composition of each of the diets is shown in Table 3.

For comparative purposes the results of this experiment are presented together with those of a similar study (Clement, 1974), in which chicks were fed diets containing 20 parts SFO, HER or LER or 15 parts HER or LER plus 5 parts palmitic or oleic acid for 7 and 26 days. The combined results of both experiments were analyzed statistically using analysis of variance. Sources of variance consisted of treatments (n=7),

TABLE 3

Fatty acid composition of oils

Fatty acid	Per cent of total fatty acids						
	SFO[1]	HER[1]	LER[1]	HER + Fatty acid		LER + Fatty acid	
				Palmitic	Oleic	Palmitic	Oleic
14:0	-	-	-	0.1	0.3	-	0.3
16:0	5.3	3.5	3.4	34.6	3.5	31.5	3.2
16:1	-	0.1	0.1	-	1.5	-	1.2
18:0	3.6	1.4	1.4	1.3	1.2	1.3	1.1
18:1	14.1	21.9	53.6	14.8	39.8	38.2	64.1
18:2	76.5	23.5	32.4	16.0	18.8	22.7	24.1
18:3	0.1	5.7	5.8	3.8	4.0	4.2	4.1
20:0	-	0.3	0.2	0.2	0.2	0.1	-
20:1	0.2	10.5	1.6	7.0	7.6	1.0	1.0
22:0	0.1	0.5	-	0.1	-	-	-
22:1	-	32.8	1.5	22.1	23.1	1.0	0.9

[1] See footnotes 3-5, Table 2.

experiments (n=2) and duplicate groups within each treatment by experiment combination. Duncan's multiple range test (Steel and Torrie, 1960) was used for comparisons between means.

Results

Data showing the average weight gains of chicks fed diets in which 20 parts of the oil or oil mixture were substituted isocalorically for glucose are summarized in Table 4. For comparative purposes data obtained by Clement (1974) are also included (Experiment 1A). Analysis of variance and application of Duncan's multiple range test (Steel and Torrie, 1960) to the combined data from both experiments showed that chicks fed diets containing 20 parts HER grew significantly slower and consumed significantly less feed than chicks fed diets containing 20 parts SFO, the effects being similar in both experiments. The data also show that chicks fed diets containing HER plus palmitic acid grew at the same rate as chicks fed diets containing HER plus oleic acid, thus indicating that increasing the saturated to unsaturated fatty acid ratio did not affect growth significantly. This finding was consistent in the two experiments.

TABLE 4

Weight gain, energy consumption and feed efficiency of chicks fed experimental diets ad libitum for 20 or 26 days

Dietary level							
Oil		Palmitic acid	Oleic acid	Exp. No.[1]	Weight gain	Kcal consumed[2]	Feed efficiency
	g	g	g		g		kcal/g gain
SFO[3]	20	-	-	1	586	2568	4.38
				1A	595	2864	4.81
					$\underline{590}^{d}$	$\underline{2716}^{c}$	$\underline{4.60}^{a}$
HER[3]	20	-	-	1	497	2238	4.50
				1A	506	2414	4.76
					$\underline{502}^{a}$	$\underline{2326}^{a}$	$\underline{4.63}^{ab}$
HER	15	5	-	1	445	2257	5.07
				1A	582	2656	4.56
					$\underline{514}^{ab}$	$\underline{2456}^{ab}$	$\underline{4.82}^{c}$
HER	15	-	5	1	486	2318	4.77
				1A	533	2516	4.72
					$\underline{510}^{ab}$	$\underline{2417}^{ab}$	$\underline{4.74}^{bc}$
LER[3]	20	-	-	1	576	2582	4.48
				1A	538	2458	4.57
					$\underline{557}^{cd}$	$\underline{2520}^{b}$	$\underline{4.53}^{a}$
LER	15	5	-	1	567	2707	4.78
				1A	572	2696	4.71
					$\underline{570}^{cd}$	$\underline{2702}^{c}$	$\underline{4.74}^{bc}$
LER	15	-	5	1	529	2442	4.61
				1A	563	2544	4.52
					$\underline{546}^{bc}$	$\underline{2493}^{b}$	$\underline{4.56}^{a}$

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[1] Experiment 1 refers to data from this experiment, experiment 1A to data obtained by Clement (1974) in a similar study of 26 days duration.

[2] Calculated using determined metabolizable energy values for the diets.

[3] See footnotes 3-5, Table 2.

[4] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P<0.05$).

Statistical analysis of the combined data also showed that chicks fed diets containing 20 parts LER consumed significantly less feed and gained less, but not significantly less, than chicks fed diets containing 20 parts SFO. Results showed that the growth promoting properties of LER varied between experiments, being equal to SFO in this experiment (Exp. 1) but significantly less than SFO in Clement's experiment (Exp. 1A). The data also show that chicks fed diets containing LER plus palmitic acid grew at a similar rate to chicks fed diets containing LER plus oleic acid, comparable results being obtained in both experiments.

Summarized in Table 5 are data showing weight gain, energy consumption, and feed efficiency of chicks pair-fed the experimental diets for 20 days in this experiment Exp. 1) and for 26 days in a similar study (Exp. 1A) conducted by Clement (1974). The results of both studies show that the addition of neither palmitic acid nor oleic acid to a diet containing HER or LER had any beneficial effect on growth when feed intakes were restricted to that of chicks fed diets containing unsupplemented HER. The reason why chicks fed diets containing HER ad libitum for 20 or 26 days grew faster than chicks fed an equicaloric amount of HER plus palmitic or oleic acid, or LER plus palmitic acid is unknown.

TABLE 5

Weight gain, energy consumption and feed efficiency of chicks pair-fed experimental diets for 20 or 26 days.

Dietary level							
Oil		Palmitic acid	Oleic acid	Exp. No.[1]	Weight gain	Kcal consumed[2]	Feed efficiency
	g	g	g		g		kcal/g gain
SFO[3]	20	-	-	1	451[4]	2042	4.53
				1A	486 $\underline{468}^{bc}$	2392 $\underline{2217}^{ab}$	4.92 $\underline{4.72}^{ab}$
HER[3]	20	-	-	1	497	2238	4.50
				1A	506 $\underline{502}^{d}$	2414 $\underline{2326}^{c}$	4.76 $\underline{4.63}^{a}$
HER	15	5	-	1	430	2235	5.20
				1A	490 $\underline{460}^{ab}$	2352 $\underline{2294}^{c}$	4.80 $\underline{5.00}^{c}$
HER	15	-	5	1	459	2301	5.01
				1A	496 $\underline{478}^{c}$	2448 $\underline{2374}^{c}$	4.94 $\underline{4.98}^{c}$
LER[3]	20	-	-	1	448	2092	4.66
				1A	482 $\underline{465}^{bc}$	2298 $\underline{2195}^{a}$	4.77 $\underline{4.72}^{ab}$
LER	15	5	-	1	416	2171	5.22
				1A	480 $\underline{448}^{a}$	2358 $\underline{2264}^{bc}$	4.91 $\underline{5.06}^{c}$
LER	15	-	5	1	428	2112	4.94
				1A	486 $\underline{457}^{ab}$	2253 $\underline{2182}^{a}$	4.64 $\underline{4.79}^{b}$

[1] Experiment 1 refers to data from this experiment, experiment 1A to data obtained by Clement (1974) in a similar study of 26 days duration.

[2] Calculated using determined metabolizable energy values for the diets.

[3] See footnotes 3-5, Table 2.

[4] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P<0.05$).

Data on carcass composition summarized in Table 6 show that chicks fed diets containing HER or LER deposited significantly less body fat and utilized energy less efficiently ($P<0.05$) than chicks fed a diet containing SFO, the effects being more marked in Experiment 1 than in Experiment 1A. Results show that chicks fed diets containing HER or LER supplemented with palmitic acid deposited similar amounts of fat and utilized energy with the same efficiency as chicks fed diets containing HER or LER supplemented with oleic acid, respectively. These results indicate that the decreased fat deposition and energetic efficiency observed in chicks fed diets containing HER or LER was not due to their low saturated to unsaturated fatty acid ratio.

Analysis of the combined results of both experiments summarized in Table 7 show that after 7 days of pair-feeding, chicks fed diets containing 20 parts HER deposited significantly less fat and utilized energy with significantly less efficiency than chicks pair-fed similar levels of SFO or LER. Modification of the fatty acid composition of either HER or LER by the addition of palmitic or oleic acid had no significant effect on the amount of fat deposited or the efficiency of energy utilization after 7 days, the results being consistent between experiments.

TABLE 6

Energy consumption, carcass gain of fat and protein, and energy utilization of chicks pair-fed experimental diets for 20 or 26 days.

	Dietary level					Carcass Gain		
Oil		Palmitic acid	Oleic acid	Exp No.[1]	Kcal consumed[2]	Fat	Protein	Energy utilization[3]
	g	g	g			g	g	
SFO[4]	20	-	-	1	2042	52.7	76.2	2.20
				1A	2392	56.3	93.6	2.26
					2217^{ab}	54.5^{c}	84.9^{d}	2.23^{a}
HER[4]	20	-	-	1	2238	46.4	74.4	2.61
				1A	2413	46.6	93.1	2.50
					2326^{c}	46.5^{a}	83.8^{bcd}	2.56^{cd}
HER	15	5	-	1	2235	48.2	71.8	2.60
				1A	2352	52.7	91.2	2.32
					2294^{c}	50.4^{b}	81.5^{ab}	2.46^{bcd}
HER	15	-	5	1	2301	46.2	76.9	2.64
				1A	2448	51.9	92.2	2.42
					2374^{c}	49.0^{ab}	84.6^{cd}	2.53^{cd}
LER	20	-	-	1	2092	48.0	73.7	2.41
				1A	2298	50.4	90.6	2.32
					2195^{a}	49.2^{ab}	82.2^{abcd}	2.36^{b}
LER	15	5	-	1	2171	45.8	71.8	2.59
				1A	2358	49.5	91.8	2.39
					2264^{bc}	47.6^{ab}	81.8^{abc}	2.49^{cd}
LER	15	-	5	1	2112	45.3	70.7	2.56
				1A	2253	51.8	90.7	2.26
					2182^{a}	48.6^{ab}	80.7^{a}	2.41^{bc}

---

[1] Experiment 1 refers to data from this experiment, experiment 1A to data obtained by Clement (1974) in a similar study of 26 days duration.

[2] Calculated using determined metabolizable energy value for the diets.

[3] Kilocalories of metabolizable energy consumed/kcalorie gained.

[4] See footnotes 3-5, Table 2.

[5] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

TABLE 7

Weight gain, energy consumption, carcass gain of fat and protein and energy utilization of chicks pair-fed experimental diets for 7days.

Oil	Dietary level						Carcass gain		
		Palmitic acid	Oleic acid	Exp. No.[1]	Weight gain	Kcal consumed[2]	Fat	Protein	Energy utilization[3]
	g	g	g		g[5]		g	g	
SFO[4]	20	-	-	1	118	489	8.8	15.6	2.86
				1A	92	410	10.7	15.5	2.18
					$\underline{105}^{a}$	$\underline{450}^{a}$	$\underline{9.8}^{c}$	$\underline{15.6}^{a}$	$\underline{2.52}^{a}$
HER[4]	20	-	-	1	117	540	7.2	15.0	3.56
				1A	99	419	7.4	16.0	2.60
					$\underline{108}^{a}$	$\underline{480}^{ab}$	$\underline{7.3}^{a}$	$\underline{15.5}^{a}$	$\underline{3.08}^{b}$
HER	15	5	-	1	107	543	6.6	14.0	3.86
				1A	95	410	8.5	15.9	2.40
					$\underline{101}^{a}$	$\underline{476}^{ab}$	$\underline{7.6}^{ab}$	$\underline{15.0}^{a}$	$\underline{3.13}^{b}$
HER	15	-	5	1	113	556	6.6	14.5	3.86
				1A	92	423	8.7	15.2	2.52
					$\underline{102}^{a}$	$\underline{490}^{b}$	$\underline{7.6}^{ab}$	$\underline{14.8}^{a}$	$\underline{3.19}^{b}$
LER[4]	20	-	-	1	115	498	7.7	14.4	3.22
				1A	99	392	10.0	16.6	2.08
					$\underline{107}^{a}$	$\underline{445}^{a}$	$\underline{8.8}^{bc}$	$\underline{15.5}^{a}$	$\underline{2.65}^{a}$
LER	15	5	-	1	122	525	8.2	14.8	3.30
				1A	96	404	10.2	16.3	2.15
					$\underline{109}^{a}$	$\underline{464}^{ab}$	$\underline{9.2}^{c}$	$\underline{15.6}^{a}$	$\underline{2.72}^{a}$
LER	15	-	5	1	114	499	8.6	14.7	3.04
				1A	99	386	11.5	16.0	1.94
					$\underline{106}^{a}$	$\underline{442}^{a}$	$\underline{10.0}^{c}$	$\underline{15.4}^{a}$	$\underline{2.49}^{a}$

---

[1] Experiment 1 refers to data obtained in this experiment, experiment 1A to data obtained by Clement (1974), in a similar study.

[2] Calculated using determined metabolizable energy values for the diets.

[3] Kilocalories of metabolizable energy consumed/kcaloric gained.

[4] See footnotes 3-5, Table 2.

[5] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different. ($P < 0.05$).

Data showing the average weight and fat content of the hearts of chicks fed diets containing 20 parts oil or oil mixture for 7 or 20 days (Exp. 1), are shown in Table 8. For comparative purposes data obtained by Clement (1974) are also included, (Exp. 1A). Analysis of variance and application of Duncan's multiple range test (Steel and Torrie, 1960) to the combined results of the two experiments showed that chicks fed diets containing 20 parts HER had significantly larger hearts at 20 or 26 days, but not at 7 days, than chicks fed diets containing 20 parts SFO or LER under either ad libitum or pair-feeding conditions. These results were observed in both experiments. Results also showed that the increase in heart size of chicks pair-fed a diet containing 20 parts HER for 20 or 26 days was not due to the accumulation of fat; the fat content of hearts of chicks fed diets containing HER, LER, or SFO were similar in both experiments. Heart size and levels of cardiac lipid in chicks fed diets containing 20 parts LER were similar to those of chicks fed diets containing 20 parts SFO, irrespective of method and length of feeding, and experiment.

Statistical analysis of the combined data for the two experiments also showed that chicks fed diets containing HER plus palmitic acid had hearts similar in size and fat content to chicks fed diets containing HER plus oleic

TABLE 8

Weight and fat content of hearts of chicks fed experimental diets for 7 and for 20 or 26 days.

Dietary level					Ad libitum		Pair-fed			
					20 or 26 day		7 day		20 or 26 day	
Oil		Palmitic acid	Oleic acid	Exp. No.[1]	Heart size[2]	Fat content[3]	Heart size[4]	Fat content	Heart size	Fat content
	g	g	g		mg/g	%	mg/g	%	mg/g	%
SFO[5]	20	-	-	1	7.12[6]	-	9.34	9.66	7.90	14.44
				1A	6.10	-	8.72	9.20	6.36	12.80
					6.61^{a}		9.03^{a}	9.43^{a}	7.13^{a}	13.62^{c}
HER[5]	20	-	-	1	7.94	-	9.85	9.83	8.80	14.62
				1A	7.20	-	9.40	7.94	7.93	12.23
					7.57^{b}		9.62^{ab}	8.88^{a}	8.36^{d}	13.42^{bc}
HER	15	5	-	1	9.12	-	9.80	8.54	8.86	13.59
				1A	6.65	-	8.99	8.80	6.82	11.59
					7.88^{b}		9.40^{ab}	8.67^{a}	7.84^{bc}	12.59^{ab}
HER	15	-	5	1	8.10	-	10.05	10.20	9.10	13.88
				1A	7.28	-	8.85	8.30	7.45	11.55
					7.69^{b}		9.45^{ab}	9.25^{a}	8.28^{cd}	12.72^{bc}
LER[5]	20	-	-	1	7.36	-	9.34	10.46	7.55	13.54
				1A	6.29	-	8.77	9.37	6.57	11.68
					6.85^{a}		9.06^{a}	9.92^{a}	7.06^{a}	12.61^{abc}
LER	15	5	-	1	7.30	-	9.26	9.84	8.26	12.98
				1A	6.16	-	9.28	9.80	6.40	10.40
					6.73^{a}		9.27^{ab}	9.82^{a}	7.33^{a}	11.69^{a}
LER	15	-	5	1	7.56	-	10.36	10.33	8.14	14.22
				1A	6.48	-	9.50	10.06	6.60	12.12
					7.02^{a}		9.93^{b}	10.20^{a}	7.37^{ab}	13.17^{bc}

[1] Experiment 1 refers to this experiment, experiment 1A to data obtained by Clement (1974) in a similar study.

[2] mg heart/g body weight.

[3] Determined on a wet weight basis.

[4] mg heart/g carcass.

[5] See footnotes 3-5, Table 2.

[6] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

acid. This effect was observed in all feeding regimens and in both experiments. Thus, these results indicate that the increase in heart size of chicks fed diets containing HER was not due to a low saturated to unsaturated fatty acid ratio. Results also showed that, irrespective of feeding regimen, and in both Experiment 1 and in Experiment 1A, chicks fed diets containing LER plus palmitic acid had hearts similar in size to chicks fed diets containing LER plus oleic acid. This finding gives support to the concept that a low saturated to unsaturated fatty acid ratio was not the cause of the enlarged hearts observed in chicks fed diets containing HER.

Data summarized in Table 9 show the erucic acid content of heart and carcass lipid of chicks pair-fed the diets containing the oils and oil mixtures. Analysis of variance of the factorial arrangement of treatments showed that chicks fed diets containing HER deposited significantly more erucic acid in heart and carcass lipid than chicks fed diets containing LER. This effect was observed in both experiments. The data also show that modification of HER by the addition of oleic acid caused significantly less accumulation of erucic acid in both heart and carcass lipid than did the addition of palmitic acid at 20 and 26 days, when the results of both experiments were combined. However, in the individual experiments, the effect was significant in Experiment 1A but

TABLE 9

Erucic acid content[1] of heart and carcass fat of chicks fed experimental diets

	Dietary level				7 day		20 or 26 day	
Oil		Palmitic acid	Oleic acid	Exp. No.[2]	Carcass	Heart	Carcass	Heart
	g	g	g		%	%	%	%
HER[3]	20	-	-	1	7.9[4]	5.8	11.4	9.1
				1A	8.9	8.4	11.8	9.5
					8.4^{a}	7.1^{b}	11.6^{a}	9.3^{bc}
HER	15	5	-	1	5.9	4.5	10.2	9.3
				1A	8.2	6.8	13.0	11.2
					7.0^{b}	5.6^{c}	11.6^{a}	10.2^{b}
HER	15	-	5	1	5.9	3.9	9.3	8.4
				1A	6.9	4.4	9.6	9.1
					6.4^{bc}	4.2^{d}	9.4^{c}	8.8^{c}
LER[3]	20	-	-	1	1.3	0.3	1.0	0.6
				1A	2.9	0.8	2.0	1.3
					2.1^{e}	0.6^{f}	1.5^{d}	1.0^{d}
LER	15	5	-	1	1.5	0.5	0.9	0.5
				1A	1.3	0.7	1.5	1.0
					1.4^{ef}	0.6^{f}	1.2^{d}	0.8^{d}
LER	15	-	5	1	1.2	0.3	0.5	0.5
				1A	1.2	0.7	1.2	0.8
					1.2^{ef}	0.5^{f}	0.8^{d}	0.6^{d}

[1] Per cent of total fatty acids.

[2] Experiment 1 refers to this experiment , experiment 1A to data obtained by Clement (1974) in a similar study.

[3] See footnotes 3-5, Table 2.

[4] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values for carcass and /or heart samples obtained for a given feeding regimen without a common letter in their superscript are significantly different ($P < 0.05$).

only a trend was observed in Experiment 1. Similar results were obtained at 7 days except that in the case of carcass lipid the decrease in erucic acid caused by the addition of oleic acid to HER was not great enough to be significant. Modification of the fatty acid composition of LER by the addition of palmitic or oleic acid had no significant effect on erucic acid deposition at either 7 or 20 and 26 days; this observation was the same in the two experiments.

The effect of modifying the fatty acid composition of HER and LER by the addition of palmitic or oleic acid on the deposition of eicosenoic acid in heart and carcass lipid is shown in Table 10. Analysis of variance of the factorial arrangement of treatments showed that modification of the fatty acid composition of HER by the addition of oleic acid resulted in significantly less accumulation of eicosenoic acid in both heart and carcass tissue than did the addition of palmitic acid, when the results of both experiments were combined. In the individual experiments, the effect was significant in Experiment 1A but only a trend was observed in Experiment 1. Modification of the fatty acid composition of LER had no significant effect on the deposition of eicosenoic acid in heart and carcass lipid.

TABLE 10

Eicosenoic acid content[1] of heart and carcass fat of chicks fed experimental diets.

Dietary level					7 day		20 or 26 day	
Oil		Palmitic acid	Oleic acid	Exp. No.[2]	Carcass	Heart	Carcass	heart
	g	g	g		%	%	%	%
HER[3]	20	-	-	1	9.5[4]	8.1	11.0	10.4
				1A	7.3	10.0	11.8	11.5
					$\underline{8.4}^{a}$	$\underline{9.0}^{a}$	$\underline{11.4}^{a}$	$\underline{11.0}^{a}$
HER	15	5	-	1	7.5	5.8	8.0	8.8
				1A	6.7	8.1	9.9	10.3
					$\underline{7.1}^{b}$	$\underline{7.0}^{b}$	$\underline{9.0}^{b}$	$\underline{9.6}^{b}$
HER	15	-	5	1	6.0	5.6	7.7	7.7
				1A	5.4	5.8	7.9	8.4
					$\underline{5.7}^{c}$	$\underline{5.7}^{c}$	$\underline{7.8}^{c}$	$\underline{8.0}^{c}$
LER[3]	20	-	-	1	4.7	1.4	2.0	1.8
				1A	2.3	2.4	2.9	2.9
					$\underline{3.5}^{d}$	$\underline{1.9}^{f}$	$\underline{2.4}^{d}$	$\underline{2.4}^{d}$
LER	15	5	-	1	3.9	1.1	1.5	1.6
				1A	1.7	2.1	2.3	2.4
					$\underline{2.8}^{de}$	$\underline{1.6}^{f}$	$\underline{1.9}^{de}$	$\underline{2.0}^{de}$
LER	15	-	5	1	3.2	1.2	0.6	1.5
				1A	1.6	2.2	2.0	2.1
					$\underline{2.4}^{ef}$	$\underline{1.7}^{f}$	$\underline{1.3}^{e}$	$\underline{1.8}^{de}$

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[1] Per cent of total fatty acids.

[2] Experiment 1 refers to this experiment, experiment 1A to data obtained by Clement (1974) in a similar study.

[3] See footnotes 3-5, Table 2.

[4] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values for carcass and /or heart samples obtained for a given feeding regimen without a common letter in their superscript are significantly different ($P < 0.05$).

Discussion

The finding that chicks fed diets containing 15 parts HER plus 5 parts palmitic acid had similar rates of growth, feed intakes, and heart size when fed ad libitum, and energetic efficiency when pair-fed, to chicks fed diets containing 15 parts HER plus 5 parts oleic acid showed that the depression in growth, reduced energetic efficiency and increased heart size observed in chicks fed diets containing HER was not due to an unbalanced saturated to monounsaturated fatty acid ratio, at least when palmitic acid was fed in the free form.

Walker (1970) observed that the addition of 5% tallow to a diet containing 20% HER significantly increased weight gain of chicks after 21 days, and attributed this effect to the presence of the saturated fatty acids, palmitic and stearic, in tallow. However, in agreement with the results of the present study, Walker (1970) found that the addition of 2.5 or 5% of a mixture of palmitic and stearic acids had no significant beneficial effect on chick growth.

In work with the rat, a beneficial effect on growth of increasing the saturated fatty acid content of diets containing HER has been demonstrated in some experiments (Beare et al., 1963), but could not be reproduced in others (Beare-Rogers et al., 1972). Furthermore,

Rocquelin et al., (1970), as a result of feeding mixtures of synthetic triglycerides of similar saturated to unsaturated fatty acid ratio to HER, have concluded that when the dietary linoleic acid supply exceeds 10% of the total fatty acid content (as in rapeseed oils), there is no growth depressing effect due to an unbalanced ratio of saturated to monounsaturated fatty acids in the diet.

The addition of saturated fatty acids to a diet containing HER has been observed to produce a variable response on chick growth. Clement (1974) observed that decreasing the level of HER in the diet from 20 to 15 parts and adding 5 parts palmitic acid significantly improved weight gain of chicks fed ad libitum and energetic efficiency of chicks pair-fed for 26 days. Since in Clement's experiment the diet containing 15 parts HER plus 5 parts palmitic acid had similar nutritive properties to a diet containing 15 parts HER plus 5 parts oleic acid, the growth promoting effects observed may have been due, at least in part, to the reduced level of HER in the diet. The reason why in contrast to Experiment 1A, reducing the level of HER from 20 to 15 parts failed to increase weight gain in Experiment 1 is not known.

That erucic acid and eicosenoic acids contained in HER contributed to the decreased growth, reduced

energetic efficiency and enlargement of the heart of chicks fed diets containing HER is shown by the finding that chicks fed diets containing 20 parts LER grew faster, utilized energy more efficiently and had smaller hearts than chicks fed comparable diets containing HER. Similarly, Vogtmann et al. (1973) observed that chicks fed diets containing 15% LER for 4 weeks grew faster than chicks fed diets containing 15% HER. In contrast, Sheppard et al. (1971) found that chicks fed diets containing 16% LER for 3 weeks grew at a rate similar to chicks fed diets containing 16% HER and significantly slower than chicks fed diets containing 16% corn oil. In studies with the rat it has been well demonstrated that erucic acid is the primary agent responsible for the growth depressing properties of HER (Abdellatif and Vles, 1973; Rocquelin et al., 1970; Thomasson and Boldingh, 1955).

Results of Experiment 1 and 1A showed that although LER had a higher nutritive value for the chick than HER, it was inferior to SFO in the efficiency with which it was utilized by the chick in promoting tissue gain. Since increasing the palmitic acid intake of chicks fed diets containing LER did not enable them to utilize energy as efficiently as chicks fed diets containing SFO, it can be concluded that some factor other than

the low saturated to unsaturated fatty acid ratio in LER was contributing to the difference. Recently, Heijkensjöld and Ernster (1975) have noted that mitochondria isolated from the hearts of rats fed diets containing as little as 1.4% erucic acid for 2 to 4 weeks oxidize substrates such as palmitylcarnitine at reduced rates. Thus they (Heijkensjöld and Ernster, 1975) suggest that erucic acid, even at low concentration, may interfere with the enzyme system involved in the mitochondrial oxidation of long-chain fatty acids. Whether low levels of erucic acid in LER contributed to the reduced energetic efficiency observed in chicks is unknown.

The reason why supplementation of a diet containing HER with oleic acid reduced the deposition of erucic and eicosenoic acid in the heart and carcass lipid significantly more than supplementation of a comparable diet with palmitic acid is unknown. One factor which may contribute to the difference is the fact that oleic acid is more completely absorbed by the chick than palmitic acid (Renner and Hill, 1961), thus reducing the concentration of erucic and eicosenoic acid presented to the heart and skeletal muscle for oxidation. Renner and Hill (1961) have reported the absorbability of oleic and palmitic acid to be 88 and -1% respectively.

EXPERIMENTS 2 AND 3

Results of Experiment 1 and those of Clement (1974) showed that chicks fed a diet containing 20 parts high or low erucic acid rapeseed oil required more energy per unit of energy gained than chicks fed a diet containing 20 parts sunflowerseed oil. The following studies were, therefore, designed to determine whether energy utilization is altered in rapeseed oil diets by dietary induced transitions in mitochondrial metabolism and concomitant alterations in metabolic conservation of energy.

Materials and Methods

Diets containing 20 parts high erucic acid rapeseed oil (HER), 20 parts low erucic acid rapeseed oil (LER), or 20 parts sunflowerseed oil (SFO) were formulated from the high carbohydrate diet (Table 1), by substitution of the test oil isocalorically for glucose. Metabolizable energy values used in formulation of the diets were; glucose 3.64 kcal/g (Hill et al., 1960), HER 7.37 kcal/g, LER 8.79 kcal/g and SFO 8.88 kcal/g (Renner, 1967). Cellulose was added to improve texture and maintain a similar caloric density between the diets

Since determination of metabolizable energy was not intended, chromic oxide mix was omitted from the diets. The composition of the diets is shown in Table 11. The fatty acid composition of the oils is shown in Table 12.

Two duplicate experiments were conducted. In each experiment, the diets were fed to duplicate groups of 10 male crossbred (Dominant White X White Plymouth Rock) chicks, from 4 to 11 and from 4 to 32 days of age. Feed intakes of chicks fed diets containing LER and SFO were restricted to that of chicks fed diets containing HER.

The method of allotment, feeding and housing of chicks has been described (Experiment 1). Data on growth and feed consumption were obtained weekly and feed wastage was determined daily.

At the end of each feeding trial chicks were killed by decapitation. Tissue was immediately excised for isolation of mitochondria as described by Dow (1967a, 1967b), and tissue analysis of fat and fatty acid composition by methods described previously (Experiment 1).

Initially 5 chicks per group were killed for isolation of mitochondria from the right ventricle of the heart. Subsequently, the remaining 5 chicks in each group were killed for isolation of mitochondria from muscle of the lower leg (peroneus longus and tibialis anterior). At the same time tissue samples of heart

TABLE 11

Composition of diets

Type of diet		Constant ingredients	Glucose[1]	Cellulose[2]	Total
Oil	Amount				
	g	g	g	g	g
SFO[2]	20	51.13	4.81	9.44	85.38
HER[3]	20	51.13	13.11	9.44	93.68
LER[4]	20	51.13	5.31	9.44	85.88

[1] Cerelose.

[2] Alpha-floc BW-40. Brown Company, Berlin, New Hampshire.

[3] Sunflowerseed oil, "Safflo." Co-op Vegetable Oils Ltd., Altona, Manitoba.

[4] High erucic acid rapeseed oil. Western Canadian Seed Processors Ltd., Lethbridge, Alberta.

[5] Low erucic acid rapeseed oil. Western Canadian Seed Processors Ltd., Lethbridge, Alberta.

TABLE 12

Fatty acid composition of oils

	Per cent of total fatty acids		
Fatty Acids	SFO[1]	HER[1]	LER[1]
14.0	0.1	0.1	0.1
16.0	6.5	2.9	4.7
16.1	0.1	0.2	0.3
18.0	5.0	1.6	2.3
18.1	15.7	33.6	45.2
18.2	70.4	19.2	31.6
18.3	0.6	4.7	8.8
20.0	0.6	0.9	0.4
20.1	0.6	11.4	3.0
22.0	1.0	0.6	0.4
22.1	1.2	24.8	3.1

[1] See footnotes 3-5, Table 11.

and leg muscle were removed and stored at -29° until analyzed for fat and constituent fatty acids.

Tissue removed for isolation of mitochondria was immediately cut into small sections and washed with several portions of chilled isolation medium containing mannitol 0.21M, sucrose 0.07M and EDTA 10 mM, to remove external blood. Subsequent isolation procedures were conducted at 0 to 5°. After tissue homogenization in a glass-teflon tissue homogenizer, the homogenate was centrifuged (500g for 5 min), filtered to remove lipids and recentrifuged (12000g for 10 min). The brown mitochondrial pellet was resuspended in isolation medium (10 mls) containing 0.5% (w/v) heparin and centrifuged (2400g for 5 min). Mitochondria were then suspended in a solution containing mannitol 0.21M, sucrose 0.07M, EDTA 10mM, Tris-HCl buffer 0.01M (pH 7.4), albumin 0.1% (w/v) and heparin 330 units/ml. Protein was measured by a colorimetric method (Lowry et al., 1951).

Mitochondrial rates of oxygen uptake and respiratory control indices were measured polarographically at 37° using a YSI oxygen monitor (Model 53, Yellow Springs Instrument Company, Ohio, USA), equipped with two Clark type oxygen sensors in the following reaction media:

pyruvate 10mM with malate 2mM; KCl 15mM; potassium phosphate 30mM; Tris-HCl, pH 7.4, 25mM; sucrose 45mM; mannitol 10mM; $MgCl_2$ 5mM; EDTA 7mM; albumin 0.2% (w/v); glucose 20mM; cytochrome C 0.015mM; NAD 0.5mM; mitochondrial protein 180-300ug; total reaction volume 2 mls. A standardized amount of ADP (220 nmoles) was added after the basal respiratory rate was determined. The state 3 respiratory rate in the presence of ADP, the state 4 respiratory rate after exhaustion of ADP, respiratory control indices and ADP/0 ratios were determined on third and subsequent cycles as described by Chance and Williams (1956). The uncoupling action of free fatty acids liberated during mitochondria isolation or incubation was prevented by the addition of albumin and EDTA.

Results

Data showing average 7 and 28 day weight gain, energy consumption, and feed efficiency of chicks pair-fed diets in which 20 parts HER, LER or SFO was substituted isocalorically for glucose in a high carbohydrate diet are summarized in Table 13. Analysis of variance and application of Duncan's multiple range test (Steel and Torrie, 1960) to the data showed that after 7 days pair-feeding no significant difference in weight gain or in feed efficiency was observed. After 28 days

TABLE 13

Weight gain, energy consumption and feed efficiency of chicks pair-fed experimental diets for 7 or 28 days

Diet	Duration of feeding	Exp. No.	Mortality	Weight gain	Energy[1] consumption	Feed efficiency
	Days		/20 chicks	g	kcal	kcal/g gain
SFO[2]	7	2	0	94[3]	429	4.54
		3	0	$100_{\underline{97}^{a}}$	$512_{\underline{470}^{a}}$	$5.13_{\underline{4.84}^{a}}$
HER[2]	7	2	0	91	458	5.03
		3	0	$98_{\underline{94}^{a}}$	$522_{\underline{490}^{a}}$	$5.33_{\underline{5.18}^{a}}$
LER[2]	7	2	0	78	402	5.15
		3	0	$92_{\underline{85}^{a}}$	$451_{\underline{427}^{b}}$	$4.90_{\underline{5.02}^{a}}$
SFO	28	2	0	590	3312	5.61
		3	1	$550_{\underline{570}^{a}}$	$3273_{\underline{3292}^{a}}$	$5.95_{\underline{5.78}^{a}}$
HER	28	2	2	614	3306	5.38
		3	5	$643_{\underline{628}^{a}}$	$3268_{\underline{3287}^{a}}$	$5.08_{\underline{5.23}^{b}}$
LER	28	2	0	594	3299	5.55
		3	1	$570_{\underline{582}^{a}}$	$3263_{\underline{3281}^{a}}$	$5.72_{\underline{5.64}^{a}}$

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[1] Calculated from calculated metabolizable energy values.

[2] See footnotes 3-5, Table 11.

[3] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

pair-feeding, however, chicks fed diets containing HER gained significantly more weight per unit of energy consumed than chicks fed diets containing SFO or LER. This finding is in contrast to results obtained previously (Experiments 1 and 1A) and may be due at least in part to loss of chicks fed diets containing HER during the last 10 days of the experimental period.

Post mortem examination of chicks fed diets containing HER for 28 days showed that 2/3 had visual signs of hydropericardium and 1/4 had abnormal livers (Table 14). Chicks fed diets containing SFO or LER for 28 days or SFO, HER or LER for 7 days did not exhibit visual signs of pathological heart or liver changes.

Hearts of chicks fed diets containing HER for 7 and 28 days were significantly heavier than hearts of chicks fed diets containing LER or SFO (Table 15), however, results showed that the increase in heart size was not due to accumulation of fat (Table 15). No significant difference in heart size or heart fat content was observed between groups fed diets containing SFO or LER. Fat content of leg muscle from chicks fed the experimental diets was similar (Table 15).

TABLE 14

Incidence of hydropericardium and abnormal liver changes in chicks pair-fed experimental diets for 7 or 28 days

Diet	Duration of feeding	Exp. No.	Hydropericardium	Abnormal liver changes[1]
	Days		incidence/total chicks	incidence/total chicks
SFO[2]	7	2	0/20	0/20
		3	0/20	0/20
HER[2]	7	2	0/20	0/20
		3	0/20	0/20
LER[2]	7	2	0/20	0/20
		3	0/19	0/19
SFO	28	2	0/20	0/20
		3	0/19	0/19
HER	28	2	11/18	3/18
		3	11/15	5/15
LER	28	2	0/20	0/20
		3	0/19	0/19

[1] Enlarged and/or discolored.

[2] See footnotes 3-5, Table 11.

TABLE 15

Weight of hearts and fat content of heart and skeletal muscle of chicks pair-fed experimental diets for 7 or 28 days.

	Duration of feeding	Exp. No.	Heart size[1]	Fat content	
				Heart	Muscle
	Days		mg/g	% wwb[2]	
SFO[3]	7	2	7.74[4]	12.42	5.18
		3	7.75 7.74^{a}	10.10 11.26^{a}	4.18 4.68^{a}
HER[3]	7	2	8.54	9.13	4.44
		3	7.96 8.25^{b}	8.38 8.76^{b}	3.50 3.97^{a}
LER[3]	7	2	7.34	11.21	4.33
		3	7.51 7.42^{a}	8.26 9.74^{ab}	4.22 4.28^{a}
SFO	28	2	6.68	16.18	3.17
		3	7.06 6.87^{a}	16.68 16.43^{a}	3.57 3.37^{a}
HER	28	2	8.98	16.84	2.65
		3	8.54 8.76^{b}	17.07 16.96^{a}	3.31 2.98^{a}
LER	28	2	6.60	15.71	3.28
		3	6.90 6.75^{a}	15.94 15.82^{a}	2.99 3.14^{a}

[1] mg heart/g body.

[2] Determined on a wet weight basis.

[3] See footnotes 3-5, Table 11.

[4] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

Summarized in Tables 16 and 17 are data showing the % saturated fatty acids, unsaturation index and % n-9 fatty acids of lipids from heart and skeletal muscle of chicks pair-fed the experimental diets for 7 and 28 days. The level of saturated fatty acids present in the tissues was consistently lower in chicks fed diets containing HER or LER than in chicks fed diets containing SFO, this difference was significant in cardiac lipid after 7 and 28 days pair-feeding but not in skeletal muscle lipid. Chicks fed diets containing HER or LER had significantly lower unsaturation indices in skeletal muscle after 28 days, and in cardiac lipid after 7 and 28 days, than chicks fed diets containing SFO. The unsaturtion indices of lipid extracted from skeletal muscle of chicks fed the experimental diets for 7 days were similar. Chicks fed diets containing SFO had a much lower ($P < 0.01$) % n-9 fatty acid level in heart and skeletal muscle lipid than chicks fed diets containing HER or LER at both 7 and 28 days. Chicks fed diets containing LER had a significantly lower % n-9 fatty acid content in cardiac lipid after 7 and 28 days and in skeletal muscle lipid after 28 days than chicks fed diets containing HER.

Oxidative activity of mitochondria isolated

TABLE 16

Per cent saturated fatty acids, unsaturation index[1] and %n-9 fatty acids of heart lipid from chicks pair-fed experimental diets for 7 or 28 days.

Diet	Duration of feeding days	Exp. No.	% saturated fatty acids	Unsaturation index	%n-9 fatty acids
SFO[2]	7	2	15.3[3]	150.5	18.8
		3	15.4 $\underline{15.4}^{c}$	151.2 $\underline{150.8}^{c}$	18.2 $\underline{18.5}^{a}$
HER[2]	7	2	14.6	121.5	53.7
		3	14.3 $\underline{14.4}^{b}$	124.4 $\underline{123.0}^{a}$	52.5 $\underline{53.1}^{c}$
LER[2]	7	2	11.8	133.1	49.1
		3	12.0 $\underline{11.9}^{a}$	133.7 $\underline{133.4}^{b}$	48.0 $\underline{48.6}^{b}$
SFO	28	2	13.7	153.2	19.2
		3	13.6 $\underline{13.6}^{b}$	154.0 $\underline{153.6}^{c}$	19.3 $\underline{19.2}^{a}$
HER	28	2	9.4	118.5	61.6
		3	9.3 $\underline{9.4}^{a}$	124.6 $\underline{121.6}^{a}$	61.5 $\underline{61.6}^{c}$
LER	28	2	9.4	133.4	52.3
		3	9.7 $\underline{9.6}^{a}$	133.7 $\underline{133.6}^{b}$	51.8 $\underline{52.0}^{b}$

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[1] Unsaturation index; sum of individual unsaturated fatty acid X number of double bonds.

[2] See footnotes 3-5, Table 11.

[3] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

TABLE 17

Per cent saturated fatty acids, unsaturation index[1] and % n-9 fatty acids of skeletal muscle lipid from chicks pair-fed experimental diets for 7 or 28 days.

Diet	Duration of feeding days	Exp. No.	%saturated fatty acids	Unsaturation index	% n-9 fatty acids
SFO[2]	7	2	16.1[3]	148.5	19.1
		3	20.0 $\underline{18.0}^{a}$	135.6 $\underline{142.0}^{a}$	24.4 $\underline{21.8}^{a}$
HER[2]	7	2	15.7	116.5	54.8
		3	16.8 $\underline{16.2}^{a}$	119.9 $\underline{118.2}^{a}$	50.6 $\underline{52.7}^{b}$
LER[2]	7	2	13.5	129.8	48.3
		3	14.6 $\underline{14.0}^{a}$	129.4 $\underline{129.6}^{a}$	46.3 $\underline{47.3}^{b}$
SFO	28	2	15.9	149.7	17.7
		3	14.0 $\underline{15.0}^{b}$	152.8 $\underline{151.2}^{c}$	19.4 $\underline{18.5}^{a}$
HER	28	2	9.1	119.6	60.5
		3	10.2 $\underline{9.6}^{a}$	124.5 $\underline{122.0}^{a}$	59.7 $\underline{60.1}^{c}$
LER	28	2	10.5	131.7	52.3
		3	11.4 $\underline{11.0}^{ab}$	135.2 $\underline{133.4}^{b}$	48.5 $\underline{50.4}^{b}$

[1] Unsaturation index; sum of individual unsaturated fatty acid X number of double bonds.

[2] See footnotes 3-5, Table 11.

[3] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

from cardiac muscle of chicks fed diets containing 20 parts SFO, HER or LER is shown in Table 18. Analysis of variance and application of Newman-Keuls comparison between ordered means (Steel and Torrie, 1960) to the combined data of Experiments 2 and 3 indicated no significant difference among the diet treatments for state 3 oxygen uptake rates, respiratory control index or rate of ATP synthesis after 7 or 28 days pair-feeding. However, ADP/O ratios were significantly lower for chicks fed diets containing HER for 28 days than for chicks fed diets containing SFO for 28 days. ADP/O ratios observed for chicks fed diets containing LER for 28 days were intermediate between and not significantly different from those observed for groups fed diets containing SFO or HER for 28 days.

Statistical analysis of the data for the individual experiments following 28 days pair-feeding showed that in Experiment 2 there were no significant differences among the diet treatments in rate of state 3 oxygen uptake, respiratory control index or rate of ATP synthesis. In Experiment 2 chicks fed diets containing 20 parts HER had significantly lower ADP/O ratios than chicks fed diets containing 20 parts SFO. Chicks fed diets containing LER had ADP/O ratios intermediate between and not significantly different from those of chicks fed diets containing SFO and HER.

TABLE 18

Oxidative activity of cardiac mitochondria isolated in the presence of heparin from chicks fed diets containing SFO, HER or LER for 7 or 28 days.

Diet	Duration of feeding	Exp. No.	Rate of state 3 oxygen uptake	Respiratory control index	ADP/O	Rate of ATP synthesis
	days		ng atom/mg protein/min			m moles/mg protein/min
SFO^{1}	7	3	1118^{2a}	2.72^{a}	2.02^{a}	2228^{a}
HER^{1}	7	3	1108^{a}	2.66^{a}	2.02^{a}	2176^{a}
LER^{1}	7	3	1020^{a}	2.05^{a}	2.08^{a}	2147^{a}
SFO	28	2	1233^{3}	2.64	1.98	2338
		3	1816	4.06	2.34	4263
			$\underline{1524}^{a}$	$\underline{3.35}^{a}$	$\underline{2.16}^{b}$	$\underline{3300}^{a}$
HER	28	2	1818	2.59	1.66	3020
		3	1589	3.35	1.78	2822
			$\underline{1704}^{a}$	$\underline{2.97}^{a}$	$\underline{1.72}^{a}$	$\underline{2921}^{a}$
LER	28	2	1209	2.81	1.76	2126
		3	1691	3.01	1.93	3216
			$\underline{1450}^{a}$	$\underline{2.91}^{a}$	$\underline{1.84}^{ab}$	$\underline{2671}^{a}$

[1] See footnotes, Table 11.

[2] Values are averages of duplicate groups for Experiment 3. Values for Experiment 2 have been omitted as chicks in this experiment were without light and feed prior to termination of the experiment. Values without a common letter in their superscript are significantly different ($P < 0.05$).

[3] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

Analysis of variance of the data for Experiment 3 showed that, as observed in Experiment 2, there were no significant differences among the diet treatments in rate of state 3 oxygen uptake and respiratory control index after 28 days pair-feeding. However, in Experiment 3 chicks fed diets containing HER or LER had significantly lower ADP/O ratios and rates of ATP synthesis than chicks fed diets containing SFO. ADP/O ratios and rates of ATP synthesis of chicks fed diets containing HER and LER for 28 days were similar.

Oxygen uptake studies of mitochondria isolated from skeletel muscle of chicks fed diets containing HER indicated significantly lower rates of state 3 oxygen uptake and ADP/O ratios after 7 days pair-feeding when compared to chicks fed diets containing SFO (Table 19). Chicks fed diets containing LER for 7 days had rates of state 3 oxygen uptake and rates of ATP synthesis which were intermediate between, and not significantly different from those of chicks fed diets containing either SFO or HER.

Mitochondria isolated from the skeletal muscle of chicks pair-fed diets containing SFO, HER or LER for 28 days had similar rates of state 3 oxygen uptake and ATP synthesis; however, ADP/O ratios of chicks fed diets containing HER were significantly lower than those of

TABLE 19

Oxidative activity of skeletel muscle mitochondria isolated in the presence of heparin from chicks fed diets containing SFO, HER or LER for 7 or 28 days.

Diet	Duration of feeding	Exp. No.	Rate of State 3 oxygen uptake	Respiratory control index	ADP/O	Rate of ATP synthesis
	days		ng atom/mg protein/min			m moles/mg protein/min
SFO[1]	7	3	577[2]^{a}	1.48^{a}	1.25^{b}	730^{a}
HER[1]	7	3	461^{b}	1.48^{a}	0.94^{a}	432^{a}
LER[1]	7	3	516^{ab}	1.84^{b}	1.30^{b}	670^{a}
SFO	28	2	475[3]	1.78	1.62	769
		3	590	1.92	2.11	1181
			$\underline{532}^{a}$	$\underline{1.85}^{a}$	$\underline{1.86}^{b}$	$\underline{975}^{a}$
HER	28	2	454	2.20	1.51	684
		3	672	2.22	1.77	1136
			$\underline{563}^{a}$	$\underline{2.21}^{b}$	$\underline{1.64}^{a}$	$\underline{910}^{a}$
LER	28	2	425	2.09	1.79	743
		3	524	1.72	1.82	956
			$\underline{474}^{a}$	$\underline{1.90}^{a}$	$\underline{1.80}^{b}$	$\underline{850}^{a}$

[1] See footnotes 3-5, Table 11.

[2] Values are averages of duplicate groups for Experiment 3. Values for Experiment 2 have been omitted as chicks were without light and feed prior to termination of the experiment. Values without a common letter in their superscript are significantly different ($P < 0.05$).

[3] Values are averages of duplicate groups. Underlined values are averages of duplicate experiments. Values without a common letter in their superscript are significantly different ($P < 0.05$).

chicks fed diets containing LER or SFO. The results show ADP/O ratios of chicks fed diets containing SFO and LER were similar. Statistical analysis showed that there were no differences between the results of Experiment 2 and Experiment 3.

Discussion

Results of these experiments with chicks indicate that the ADP/O ratios of mitochondria isolated from skeletal muscle after 7 and 28 days, and cardiac muscle after 28 days of consuming a diet containing 20 parts HER were lower than when diets containing 20 parts SFO were consumed. These results suggest that the reduced energetic efficiency (kcal consumed/kcal gained) observed for chicks fed diets containing 20 parts HER (Experiments 1 and 1A, Tables 6 and 7), may be the sequel of impaired mitochondrial function with respect to metabolic energy conservation. Thus, although chicks may consume the same amount of energy from diets containing HER, fat gain and energetic efficiency are lower than for chicks fed diets containing SFO.

No significant differences in the averages of pooled ADP/O ratios or rates of ATP synthesis were observed between mitochondria isolated from the heart and skeletal muscle of chicks fed diets containing LER and chicks fed diets containing SFO, although values

for LER were lower after 28 days. Thus the effect on chick mitochondria of feeding diets containing LER remains to be clarified. In this regard it should be noted that in vivo estimation of energetic efficiency (kcal consumed/kcal gained) showed LER to be utilized less efficiency than SFO, but more efficiently than HER. The possibility exists that erucic acid, even at the very low levels present in diets containing LER, may have contributed to the reduced fat deposition and energetic efficiency observed in chicks fed diets containing 20 parts LER in Experiments 1 and 1A (Table 6). In studies with the rat, Heijkensjöld and Ernster (1975) have reported that diets containing as little as 1.4% erucic acid caused a marked reduction in the rate at which isolated heart mitochondria oxidized such substrates as palmitylcarnitine, but had little effect on the rate of oxidation of Krebs-cycle intermediates or glutamate. Thus, Heijkensjöld and Ernster (1975) have suggested that the primary effect of erucic acid may be interference with the enzyme system involved in the mitochondrial oxidation of long-chain fatty acids, with subsequent secondary effects on mitochondrial respiration and energy supply to the heart.

The finding that mitochondria isolated from cardiac and skeletal muscle of chicks fed diets containing HER

showed reduced ADP/O ratios, but similar rates of oxygen consumption and ATP synthesis when compared with chicks fed diets containing SFO is in contrast to some results reported for the rat. Thus, Houtsmuller et al. (1970) found that after 3 days feeding, cardiac mitochondria from rats fed diets containing HER had decreased rates of oxygen uptake and ATP synthesis, but only slightly reduced ADP/O ratios, when compared with control rats fed diets containing SFO. On the other hand, Dow-Walsh et al. (1975) have reported no significant differences in rates of oxygen uptake or ATP synthesis of cardiac mitochondria freshly isolated in the presence of heparin from rats fed diets containing 20% by weight of either corn oil or HER. Clandinin (1976a) has observed reductions in substrate flux, as well as reduced ADP/O ratios and respiratory control for mitochondria isolated from rats fed HER. In these studies only prolonged feeding of LER resulted in statistically significant alterations in efficiency of oxidative phosphorylation.

The effects on chicks and rats of feeding diets containing HER has been shown to vary in other respects. Thus, results of the present experiments and those of others (Clement, 1974; Vogtmann et al., 1974) have shown that unlike the rat (Abdellatif and Vles, 1970a, 1970b; Houtsmuller et al., 1970; Kramer, 1973) the

chick is not subject to either an early rapid rise in heart lipid, or to accumulation of erucic acid in heart lipid. Studies have also shown that unlike the rat (Abdellatif and Vles, 1970a) the chick does not accumulate lipid in skeletal muscles when fed diets containing HER.

Explanation for the physical, pathological and biochemical effects of rapeseed oil feeding on rats has been sought by many researchers. Heijkenskjöld and Ernster (1975), as a result of in vivo and in vitro experiments on the effects of erucic acid on the oxidative metabolism of rat-heart mitochondria, have suggested that erucic acid causes inhibition of the initial step of fatty acid oxidation, at the acyl CoA dehydrogenase level. Such a specific inhibitory effect by erucic acid could account for the fat accumulation observed in the hearts of rats fed a diet containing HER (Abdellatif and Vles, 1970a, 1970b; Houtsmuller et al., 1970; Hornstra, 1972; Kramer et al. 1973), as well as for less specific inhibitory effects on mitochondrial respiration and thus energy supply. Similar observations have been made by Christopherson and Christiansen (1975) who, in agreement with previous studies (Christopherson and Bremer, 1972; Heijkenskjöld and Ernster, 1975) noted that the rate of oxidation of

erucylcarnitine was much slower than that of palmitylcarnitine, and that the presence of erucylcarnitine caused a significant inhibition of the mitochondrial oxidation of palmitylcarnitine. Christopherson and Christiansen (1975) have suggested that sequestration of free CoA in the form of slowly metabolized erucyl CoA may be partially responsible for the observed inhibitory effects of erucylcarnitine on the oxidation of CoA dependent substrates. Thus, in support of earlier work by Christopherson and Bremer (1972), these workers propose that the accumulation of triacylglycerols in the hearts of rats fed diets containing HER may be caused by both an inhibitory effect of erucic acid on the mitochondrial oxidation of other fatty acids and by the low rate of oxidation of erucyl CoA in the mitochondria.

In contrast, studies by Swarrtouw (1974) and Cheng and Pande (1975), have demonstrated that erucic acid does not interfere with the β-oxidation of other fatty acids by rat heart mitochondria, but rather erucic acid is itself more slowly oxidized. Thus, increased deposition of triglyceride has been attributed to increased triglyceride synthesis due to slower oxidation of erucic acid and also a simultaneous inhibition of lipolysis due to the presence of free erucic acid (Cheng and

Pande, 1975).

Failure to activate erucic acid has been proposed as the rate limiting step in the overall metabolism of erucic acid by rat heart (Kramer, 1973; Blond et al., 1975; Cheng and Pande, 1975). More recently Korsrud et al., (1976), have measured rates of acyl CoA dehydrogenase reactions in the β-oxidation of long chain fatty acids by rat-heart mitochondria. These workers suggest that the activity of acyl CoA dehydrogenase decreases with increasing fatty acid chain length, and further go on to suggest that reduced activity of acyl CoA dehydrogenase with erucic acid could be responsible for the accumulation of lipid in the heart of rats fed a diets containing HER. Earlier work by Bulhak-Jachymczyk and Hübner-Wozniak (1974), has indicated difficulty in the carnitine dependent transport of erucic acid to the site of β-oxidation.

Changes in the mitochondrial membrane composition by dietary fatty acids has also been proposed as the cause of impaired energy metabolism in animals fed diets containing HER, (Clandinin, 1976b). Blomstrand and Svensson (1974, 1975), have demonstrated that in rats, erucic acid is incorporated into mitochondrial cardiolipin. Furthermore, as the erucic acid content in the membrane increases there is a corresponding

decrease in the linoleic acid content of cardiolipin. The molecular structure of component fatty acids in the lipid molecules of the mitochondrial inner membrane influence the physical properties of this membrane (O'Brien, 1967). Thus it is suggested that the specific inhibitory effect of erucic acid on mitochondrial metabolism is related to dietary induced transitions in mitochondrial membrane fatty acid composition, thereby causing site specific alterations in molecular structure of membrane lipids known to interact with mitochondrial proteins involved in oxidative phosphorylation.

Although extensively studied the causes of the short-term, initial lipid accumulation in the hearts of rats fed diets containing HER, and of the long term lesions in the hearts of rats fed diets containing HER or LER (Beare-Rogers et al., 1974; Charlton et al., 1975; Rocquelin et al., 1970, 1971), have not been clearly defined. Similarly the cause of decreased efficiency in the usage of digested calories by rats fed diets containing HER (Hornstra, 1972), are not known. Whilst decreased rate of oxidation of substrates by mitochondria isolated from the heart of rats fed diets containing HER and LER has been demonstrated by some workers (Houtsmuller, 1970; Heijkensköld and Ernster, 1975;

Clandinin, 1976a), other workers have failed to show this (Dow-Walsh et al., 1975).

Since incorporation of HER in the diet of the chick does not result in accumulation of lipid in cardiac and skeletal muscle, the aforementioned factors proposed as contributing to lipid accumulation in cardiac muscle of rats fed diets containing HER may or may not have contributed to the decreased energetic efficiency observed both in vivo and in vitro in the chick. The reason(s) for the decreased efficiency of utilization of substrate by mitochondria when chicks are fed diets containing HER remains to be elucidated.

SUMMARY

1. Chicks fed diets containing HER supplemented with palmitic acid for 3 to 4 weeks grew at the same rate, deposited similar amounts of fat, utilized energy as efficiently and had similar heart size as chicks fed diets containing HER supplemented with oleic acid. These results show that the depression in growth, reduced fat deposition, decreased energetic efficiency, and enlarged hearts observed in chicks fed diets containing HER was not due to a low content of saturated fatty acids in the diet.

2. Chicks fed diets containing LER for 3 to 4 weeks grew faster, utilized energy more efficiently, and had smaller hearts than chicks fed diets containing HER. These results show that the erucic and eicosenoic acids contained in HER contributed to the decreased growth, reduced energetic efficiency, and enlarged hearts of chicks fed diets containing HER.

3. Chicks pair-fed diets containing 20 parts LER for 3 to 4 weeks deposited less fat and utilized energy with less efficiency than chicks pair-fed comparable diets containing SFO for 3 to 4 weeks. Since increasing the palmitic acid intake of chicks fed diets containing LER did not enable them to utilize energy with any

greater efficiency it can be concluded that some factor other than the low saturated to unsaturated fatty acid ratio was contributing to the difference.

4. Supplementation of a diet containing HER with oleic acid reduced the deposition of erucic acid and eicosenoic acid in heart and carcass lipid significantly more than supplementation of a comparable diet with palmitic acid, when the diets were fed for either 7 days or 3 to 4 weeks.

5. Mitochondria isolated from cardiac and skeletal muscle of chicks fed diets containing HER for 28 days showed similar rates of oxygen consumption and ATP synthesis when compared with mitochondria isolated from chicks fed diets containing SFO. However, ADP/O ratios of mitochondria isolated from chick cardiac and skeletal muscle after 28 days of consuming a diet containing 20 parts HER were lower than when 20 parts SFO were consumed. These results suggest that uncoupling of oxidative phosphorylation may have contributed to the decreased fat deposition and reduced energetic efficiency (kcal consumed/kcal gained) observed for chicks fed diets containing 20 parts HER.

6. Mitochondria isolated from cardiac and skeletal muscle of chicks fed diets containing LER for 28 days showed similar rates of oxygen consumption

and ATP synthesis to mitochondria isolated from cardiac and skeletal muscle of chicks fed comparable diets containing SFO. ADP/O ratios of mitochondria isolated from cardiac and skeletal muscle of chicks fed diets containing LER for 28 days were found to be lower, but not significantly lower, than when diets containing SFO were consumed. Thus, whether uncoupling of oxidative phosphorylation contributes to the decreased fat deposition and energetic efficiency (kcal consumed/kcal gained) of chicks fed diets containing LER remains to be established.

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APPENDIX

Fatty acid composition of heart and carcass lipid of chicks pair-fed experimental diets for 7 days (Exp.1).

Fatty acid		Per cent of total fatty acids						
					HER +		LER +	
		SFO[1]	HER[2]	LER[3]	Palmitic acid	Oleic acid	Palmitic acid	Oleic acid
14:0	Carcass	0.8	0.8	0.7	0.8	0.8	0.7	0.8
	Heart	0.2	0.2	0.2	0.3	0.6	0.3	0.4
16:0	Carcass	16.6	16.4	15.8	26.9	13.1	21.8	11.2
	Heart	7.2	8.9	6.7	17.8	8.4	12.4	6.1
16:1	Carcass	3.4	3.1	2.5	3.2	3.6	2.2	3.3
	Heart	1.2	2.2	1.6	2.6	3.3	1.8	2.5
18:0	Carcass	7.5	5.6	5.1	6.0	4.4	5.0	4.0
	Heart	4.8	3.8	3.4	4.9	3.8	3.5	3.2
18:1	Carcass	20.9	35.7	51.3	31.4	45.4	47.0	57.6
	Heart	18.1	34.0	50.0	33.7	46.6	45.6	57.1
18:2	Carcass	47.2	15.9	16.1	14.6	18.2	15.4	15.3
	Heart	65.7	29.7	30.6	24.8	22.0	29.5	24.5
18:3	Carcass	0.3	4.1	1.5	2.8	1.9	1.8	2.0
	Heart	1.2	6.4	4.9	4.6	4.8	4.9	4.2
20:0	Carcass	0.2	0.4	0.5	0.3	0.3	0.3	0.4
	Heart	-		-	-	-	-	-
20:1	Carcass	2.3	9.5	4.7	7.5	6.0	3.9	3.8
	Heart	0.6	8.1	1.4	5.8	5.6	1.1	1.2
22:0	Carcass	0.2	0.5	0.4	0.5	0.4	0.4	0.3
	Heart	0.9	0.8	0.9	0.9	1.0	0.4	0.6
22:1	Carcass	0.6	7.9	1.3	6.0	5.9	1.5	1.3
	Heart	0.1	5.8	0.3	4.5	3.9	0.5	0.3

[1] Sunflowerseed oil.

[2] High erucic acid rapseed oil.

[3] Low erucic acid rapeseed oil.

Fatty acid composition of heart and carcass lipid of chicks pair-fed experimental diets for 20 days (Exp.1).

Fatty Acid		Per cent of total fatty acids						
					HER +		LER +	
		SFO[1]	HER[2]	LER[3]	Palmitic acid	Oleic acid	Palmitic acid	Oleic acid
14:0	Carcass	0.2	0.4	0.4	0.5	0.5	0.5	0.3
	Heart	0.1	0.5	0.2	0.2	0.4	0.2	0.4
16:0	Carcass	8.4	8.7	7.5	17.6	7.0	18.1	5.0
	Heart	5.7	5.8	4.4	15.0	5.2	13.3	4.8
16:1	Carcass	0.5	1.7	1.2	1.9	2.7	1.8	1.6
	Heart	0.4	1.5	0.7	1.3	2.2	1.0	1.9
18:0	Carcass	5.1	3.6	3.3	3.1	3.0	3.8	1.8
	Heart	4.5	3.5	3.0	3.5	3.1	2.5	2.7
18:1	Carcass	20.1	39.9	62.3	37.6	52.2	53.3	67.7
	Heart	15.7	34.7	53.7	32.8	47.0	48.0	57.9
18:2	Carcass	64.6	20.8	20.3	18.7	15.5	18.3	20.6
	Heart	71.7	27.4	30.1	23.7	20.6	27.7	25.3
18:3	Carcass	0.1	2.5	1.9	2.4	2.2	1.7	1.8
	Heart	0.5	5.8	4.4	4.3	3.8	4.3	3.9
20:0	Carcass	-	-	-	-	-	-	-
	Heart	0.2	0.3	0.2	0.4	0.5	0.2	0.2
20:1	Carcass	0.3	11.0	2.0	8.0	7.7	1.5	0.6
	Heart	0.4	10.4	1.8	8.8	7.7	1.6	1.5
22:0	Carcass	-	-	-	-	-	-	-
	Heart	0.7	1.0	0.8	0.8	1.1	0.7	0.9
22:1	Carcass	0.8	11.4	1.0	10.2	9.3	0.9	0.5
	Heart	0.1	9.1	0.6	9.3	8.4	0.5	0.4

[1] Sunflowerseed oil.

[2] High erucic acid rapeseed oil.

[3] Low erucic acid rapeseed oil.

Fatty acid composition of heart and skeletal muscle lipid of chicks pair-fed experimental diets for 7 days (Exp. 2).

Fatty acids		Per cent of total fatty acids		
		SFO[1]	HER[2]	LER[3]
14:0	Skeletal muscle	0.2	0.4	0.3
	Heart	0.2	0.4	0.3
16:0	Skeletal muscle	10.0	10.1	8.6
	Heart	8.9	9.0	7.2
16:1	Skeletal muscle	0.8	1.6	1.1
	Heart	0.8	1.6	0.9
18:0	Skeletal muscle	5.7	4.7	4.2
	Heart	5.7	4.6	3.9
18:1	Skeletal muscle	18.4	42.6	45.4
	Heart	17.8	40.8	46.9
18:2	Skeletal muscle	63.4	23.6	31.2
	Heart	64.7	24.1	31.5
18:3	Skeletal muscle	0.6	4.3	6.0
	Heart	0.5	6.0	6.7
20:0	Skeletal muscle	0.1	0.4	0.3
	Heart	0.3	0.5	0.3
20:1	Skeletal muscle	0.4	6.6	1.8
	Heart	0.5	7.9	1.7
22:0	Skeletal muscle	0.1	0.1	0.1
	Heart	0.2	0.1	0.1
22:1	Skeletal muscle	0.3	5.6	1.1
	Heart	0.5	5.0	0.5

[1] Sunflowerseed oil.

[2] High erucic acid rapeseed oil.

[3] Low erucic acid rapeseed oil.

Fatty acid composition of heart and skeletal muscle lipid of chicks pair-fed experimental diets for 28 days (Exp.2).

Fatty acid		Per cent of total fatty acids		
		SFO[1]	HER[2]	LER[3]
14:0	Skeletal muscle	0.2	0.2	0.2
	Heart	0.1	0.2	0.1
16:0	Skeletal muscle	9.0	5.5	6.0
	Heart	7.5	5.4	5.3
16:1	Skeletal muscle	1.0	1.5	1.2
	Heart	0.4	1.1	0.7
18:0	Skeletal muscle	6.2	2.9	3.6
	Heart	5.8	3.2	3.5
18:1	Skeletal muscle	16.8	44.5	47.8
	Heart	18.1	44.3	48.8
18:2	Skeletal muscle	64.5	22.9	29.6
	Heart	66.1	22.2	31.4
18:3	Skeletal muscle	0.8	6.2	6.9
	Heart	0.6	5.9	6.2
20:0	Skeletal muscle	0.3	0.4	0.5
	Heart	0.2	0.5	0.4
20:1	Skeletal muscle	0.5	9.2	2.8
	Heart	0.7	10.8	2.5
22:0	Skeletal muscle	0.2	0.1	0.2
	Heart	0.1	0.1	0.1
22:1	Skeletal muscle	0.4	6.8	1.3
	Heart	0.4	6.5	1.0

[1] Sunflowerseed oil.

[2] High erucic acid rapeseed oil.

[3] Low erucic acid rapeseed oil.

Fatty acid composition of heart and skeletal muscle lipid of chicks pair-fed experimental diets for 7 days (Exp. 3).

		Per cent of total fatty acids		
Fatty acid		SFO[1]	HER[2]	LER[3]
14:0	Skeletal muscle	0.4	0.4	0.4
	Heart	0.2	0.4	0.2
16:0	Skeletal muscle	11.8	11.4	9.2
	Heart	9.0	9.2	7.2
16:1	Skeletal muscle	1.1	2.1	1.4
	Heart	0.7	1.8	1.2
18:0	Skeletal muscle	7.0	4.6	4.5
	Heart	5.8	4.0	4.1
18:1	Skeletal muscle	23.3	36.6	43.9
	Heart	17.4	37.8	45.1
18:2	Skeletal muscle	53.7	23.8	30.8
	Heart	64.8	23.8	31.9
18:3	Skeletal muscle	0.9	6.6	6.7
	Heart	0.9	7.5	6.9
20:0	Skeletal muscle	0.3	0.3	0.4
	Heart	0.3	0.6	0.4
20:1	Skeletal muscle	0.7	7.6	1.7
	Heart	0.6	9.1	2.2
22:0	Skeletal muscle	0.5	0.1	0.1
	Heart	0.1	0.1	0.1
22:1	Skeletal muscle	0.4	6.4	0.7
	Heart	0.2	5.6	0.7

[1] Sunflowerseed oil.

[2] High erucic acid rapeseed oil.

[3] Low erucic acid rapeseed oil.

Fatty acid composition of heart and skeletal muscle lipid of chicks pair-fed experimental diets for 28 days (Exp. 3).

Fatty acid		Per cent of total fatty acids		
		SFO[1]	HER[2]	LER[3]
14:0	Skeletal muscle	0.2	0.2	0.4
	Heart	0.1	0.2	0.2
16:0	Skeletal muscle	7.6	5.9	6.5
	Heart	7.2	5.2	5.4
16:1	Skeletal muscle	0.7	1.4	1.3
	Heart	0.5	1.0	0.7
18:0	Skeletal muscle	5.8	3.6	4.0
	Heart	5.9	3.4	3.6
18:1	Skeletal muscle	18.6	42.0	44.5
	Heart	18.3	44.4	49.0
18:2	Skeletal muscle	65.3	22.4	31.3
	Heart	66.2	22.5	32.5
18:3	Skeletal muscle	0.7	6.2	7.6
	Heart	0.6	5.7	5.4
20:0	Skeletal muscle	0.2	0.4	0.4
	Heart	0.2	0.4	0.3
20:1	Skeletal muscle	0.4	10.0	2.7
	Heart	0.6	10.5	2.1
22:0	Skeletal muscle	0.2	0.1	0.1
	Heart	0.2	0.1	0.2
22:1	Skeletal muscle	0.4	7.7	1.3
	Heart	0.4	6.6	0.7

[1] Sunflowerseed oil.

[2] High erucic acid rapeseed oil.

[3] Low erucic acid rapeseed oil.

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